Vestibular Function and Clinical Presentation of Dizziness: Are They Similar in Patients With Different Types of Migraine?

*Aline Emer Faim, †Gabriela Ferreira Carvalho, ‡Debora Bevilaqua Grossi, §Fabiola Dach, ||Andreia Ardevino Oliveira, and *Camila de Giacomo Carneiro

**Faculdade de Medicina de Ribeirão Preto, Ribeirão Preto, Brazil; †Universität zu Lübeck, Deutschland, Lübeck, Germany; ‡Departamento de Ciências da Saúde - Faculdade de Medicina de Ribeirão Preto, Ribeirão Preto, São Paulo - Brazil; §Departamento de Neurociências - Faculdade de Medicina de Ribeirão Preto, Ribeirão Preto, São Paulo - Brazil; ||Departamento de Otorrinolaringologia - Hospital das Clínicas de Ribeirão Preto, Ribeirão Preto, São Paulo - Brazil*

Objective: To compare the vestibular function and clinical aspects (vestibular and migraine symptoms) of patients divided into three groups—migraine without aura, migraine with aura, and chronic migraine—and a control group by using electronystagmography and a design questionnaire.
Study design: Case-control study.
Setting: Tertiary referral center.
Patients: Women aged between 18 and 55 years diagnosed with migraine with aura, migraine without aura, or chronic migraine according to the International Classification of Headache Disorders ICHD—third edition; diagnosis was made by a headache specialist. The control group consisted of patients' family members and hospital employees without a personal history of headache.
Main Outcome Measures: Application of a questionnaire regarding vestibular symptoms and their relation to migraine aspects. Assessment of the vestibular function by electronystagmography.
Results: This study evaluated 120 female patients. Dizziness was the most prevalent vestibular symptom in all the migraine groups, with higher prevalence in the episodic migraine with aura and chronic migraine groups. Phonophobia and photophobia during vestibular symptoms also had greater prevalence in the latter groups. Electronystagmography tests did not reveal differences among the groups, but clinical stratification showed that tests with mixed etiology abnormalities were more prevalent in the episodic migraine with aura and chronic migraine groups.
Conclusion: The prevalence of vestibular symptoms in the migraine groups and the etiology of vestibular impairment highlight that migraine affects the vestibular system. Our findings suggest that symptom progression and vestibular impact are related to migraine chronicity and presence of aura.
Key Words: Dizziness—Migraine—Vertigo—Vestibular testing.

Otol Neurotol **43:**1257–1263, 2022.

INTRODUCTION

Migraine is one of the most prevalent neurological disorders. It affects 14.7% of the global population, and its nonfatal but disabling pain conditions significantly impact patients with this disorder (1). Migraine prevalence depends on geography, socioeconomic status, race, age, and gender, and this disorder causes public health systems to incur great expenses worldwide (1,2).

Several studies have reported the cooccurrence of migraine and vestibular symptoms, which indicates that migraine is related to changes in sensory systems involved in balance or postural control, leading to imbalance and other vestibular symptoms (3,4). Migraine may affect how sensory information contributes to postural control, increasing vestibular information and reducing visual information, which highlight the relationship between migraine and vestibular symptoms (4). Migraine is also significantly associated with other diseases of the craniocervical system (5); cervical dysfunctions (6); symptoms, such as allodynia or hyperalgesia (7,8), and postural changes (9).

Numerous studies have used the altered multisensory processing theory to explain the vestibular migraine pathogenesis, but other theories have indicated that neuropeptide release may be involved in this process (3,10–15). The main mechanism of migraine theory proposes that a spreading cortical depression causes the release of neuropeptides from trigeminal ganglion, leading to a state of inflammation of the meninges and the activation of nociceptor in the brain. These neuroactive peptides participating in the cortical depression mechanism observed in migraine with aura may result in vertigo lasting minutes or hours (16), indicating that episodic migraine itself can lead to vestibular symptoms. In addition, the theory of calcitonin gene-related peptide indicates a path for vestibular impairment in vestibular migraine (17), highlighting

Address correspondence and reprint requests to Aline Faim, Faculdade de Medicina de Ribeirão Preto, Av Bandeirantes, 3900, USP Campus, Ribeirão Preto, SP, Brazil; E-mail: aline.faim@usp.br
Sources of support and disclosure of funding: FAPESP Foundation (grant 2015/18031-5).
The authors disclose no conflicts of interest.
DOI: 10.1097/MAO.0000000000003699

that the pathways underlying the types of migraine are complex and proposes that may lead to distinct phenotypes.

Although vestibular migraine has been more accurately described since 2012 (in Consensus Report of the Bárány Society and the International Headache Society), its diagnosis is challenging (18). Patients with migraine may experience variable vestibular symptoms. Indeed, studies have shown migraine subtypes related to different prevalence of vestibular symptoms, which suggests a broad spectrum of clinical symptomatology in vestibular migraine patients, causing the pathology or disease to be underdiagnosed (3,14,18,19).

However, the presence of aura and chronic migraine has been established to impact the presence and intensity of comorbidities, including vertigo (14,19) and impaired motor function (7) and motor control (20–22). The presence of aura can also intensify vestibular changes: patients experience more intense vestibular symptoms because of the changes in the oculomotor function (23).

Therefore, investigating vestibular symptoms in patients with migraine is needed for better understanding this relationship, the impact of migraine chronicity, and the presence of aura. Such investigation is also essential for managing this disorder and ensuring its effective treatment. Even though some studies have shown abnormalities in the vestibular function of migraine patients, we aim to compare the vestibular function in migraine subtypes (episodic with aura, episodic without aura, and chronic) and to investigate the spectra of this disorder.

MATERIALS AND METHODS

In this case-control study, women were evaluated in a tertiary hospital. The study was approved by the Institutional Research Ethics Committee (protocol number 15572/2016), and a written informed consent was obtained from all the participants.

A headache specialist diagnosed the patients according to the International Classification of Headache Disorders ICHD—third edition (24). The patients were stratified into three groups: episodic migraine without aura (MoA), episodic migraine with aura (MA), and chronic migraine (CM). CM comprised patients who had had headache for at least 15 days within a month in the previous 6 months and for whom the headache fulfilled the migraine criteria for at least eight of these days. Patients who had had headache for 1 to 12 days within a month in the previous 6 months were classified as MoA or MA. Patients with aura were excluded from CM. A control group (CG) consisting of patients' family members and hospital employees without headache was also included. To be part of CG, the participant should not have had a history or episode of headache in the past 10 years.

All the groups were constituted by women aged between 18 and 55 years; for CG, the participants were matched by age. The exclusion criteria adopted for all the participants (MoA, MA, CM, and CG) included any systemic disease with rheumatic or metabolic etiology; non-controlled hypertension; history of heart attack or stroke; other types of migraine, neurologic diseases, or types of headache other than migraine; use of any migraine prophylactic medications; presence of brainstem aura; any musculoskeletal impairment that could affect balance; smoking or alcoholism in the past 10 years; body mass index over 30 kg/m^2; presence of migraine attack during the interview; pregnancy; and self-reported or history of vestibular disease or use of any medication for dizziness (meclizine, flunarizine, cinnarizine, betahistine, or benzodiazepines).

A structured questionnaire to describe vestibular symptoms and their relation to migraine aspects was used to interview the patients. Information on vestibular symptoms included the presence or absence of symptoms during and between migraine attacks. The symptoms were classified according to the Bárány Society's Classification of Vestibular Symptoms (8) and included vertigo (including spontaneous internal vertigo and/or external vertigo—a sensation of distorted self-motion or false sensation of continuous visual flow), dizziness (sensation of disturbed spatial orientation without a false sense of motion), head-motion vertigo or dizziness (occurring only during head motion), and positional vertigo or dizziness (triggered by and occurring after a change of head position in space relative to gravity). The questionnaire also covered the onset of headache (years since the first episode) and the severity of migraine estimated with a rating pain scale: zero meant no headache; 10 corresponded to pain as bad as it could be.

Furthermore, information regarding the presence of headache, photophobia, and phonophobia during vestibular symptoms was assessed separately and classified as: always—vestibular symptoms always occur in the presence of migraine symptoms; occasionally—vestibular symptoms may occur in the presence or absence of migraine symptoms; never—vestibular symptoms never occur in the presence of migraine symptoms.

The Dizziness Handicap Inventory (DHI) was filled in to evaluate the impact of vestibular symptoms (25): the patients scored 25 questions, and the result was classified as mild (score between 0 and 30), moderate (31–60), or severe (61–100).

The vestibular function was evaluated through electronystagmography (ENG), which involved oculomotor assessment, rotatory chair test, and caloric test. The examination started with the oculomotor assessment, which included evaluating the presence of spontaneous nystagmus with eyes open and closed, the presence of gaze nystagmus under a fixed light (placed on the right side followed by the left side), and smooth pursuit (tracking eye movements on a pendulum-moving light), as well as an optokinetic test. Then, the rotatory chair test was performed and included sinusoidal harmonic downward movement (decreasing pendular rotatory test). First, the test was carried out with the patient with eyes open. After that, the test was performed with the patient with eyes closed. In the latter case, the presence of a response was assessed; whether the response was symmetric or asymmetric was evaluated. In addition, whether there was variation with eyes open and closed was verified (26). Finally, the caloric test was performed according to the Fitzgerald and Hallpike technique (22). The test was carried out with air stimulation (warm irrigation at 50°C and cool irrigation at 24°C) in the following order: the left ear then the right ear was submitted to warm irrigation; the right ear then the left ear was submitted to cold irrigation. The patients were instructed to remain with their eyes closed during and after irrigation, so that the presence of nystagmus could be evaluated after stimulation. Directional preponderance (DP) and canal paresis (CP) was determined by using the Jongkees method (27): DP and CP were considered abnormal when they were greater than 19% and 17%, respectively. The slow phase velocity was considered normal when the values lay between 3 and 51°/s for each response.

At the first evaluation, the patients were stratified into MoA, MA, and CM and submitted to a neurological examination, followed by application of the DHI questionnaire. The second evaluation, performed by a neurotologist, was scheduled for the morning period, after a good night's sleep, in a headache-free period, and it consisted of clinical evaluation and application of a questionnaire regarding vestibular symptoms, followed by the ENG tests (each step was standardized and performed in the same sequence).

According to the abnormalities found in each test and from a clinical perspective, the ENG test results were classified as normal, with changes of peripheral etiology, with changes of central etiology, or with changes of mixed etiology (central and peripheral).

TABLE 1. *Migraine onset and pain scale, vestibular symptoms, and DHI classification among subjects in the CG, MoA group, MA group, and CM group*

	CG (n = 30)	MoA (n = 30)	MA (n = 30)	CM (n = 30)	*p*
Migraine					
Years since onset of first headache	0	15 ± 8	18 ± 9	18 ± 11	0.675
Headache pain scale (average)	0	7.5 ± 1	7.6 ± 2	8.1 ± 2	0.217
Vestibular symptoms					
Classification					
Absence of vestibular symptoms	27 (90%)	10 (33.3%)	2 (6.7%)	4 (13.3%)	0.000*
Vertigo	1 (3.3%)	7 (23.3%)	9 (30%)	12 (40%)	
Dizziness	1 (3.3%)	9 (30%)	11 (36.7%)	13 (43.3%)	
Head-motion vertigo or dizziness	0	4 (13.3%)	6 (20%)	1 (3.3%)	
Positional vertigo or dizziness	1 (3.3%)	0	2 (6.7%)	0	
DHI					
Mild	3 (10.7%)	10 (33.3%)	5 (16.7%)	9 (30%)	0.451
Moderate	0	7 (23.3%)	16 (53.3%)	11 (36.7%)	
Severe	0	2 (6.7%)	5 (16.7%)	4 (13.3%)	

Table entries are mean ± standard deviation or total (percentage).
*Significant *p* value.
CG indicates control group; CM, chronic migraine; DHI, Dizziness Handicap Inventory; MA, migraine with aura; MoA, migraine without aura.

A χ^2 test was used to analyze the presence and subtypes of vestibular symptoms in the sample during and between migraine attacks. A Fisher's exact test was used to compare the vestibular function among the groups.

RESULTS

A total of 120 women were included in the study and equally distributed among the four groups (MoA, MA, CM, and CG; 30 in each group). The groups did not differ in terms of age (mean age = 32 years). Regarding the years since the onset of the first headache, the history of symptoms in MoA, MA, and CM did not differ significantly ($p = 0.675$). On average, the onset of symptoms occurred 3 years earlier in MA and CM than in MoA (Table 1). Headache pain severity did not differ among MoA, MA, and CM, either ($p = 0.217$) (Table 1).

Regarding the classification of vestibular symptoms, dizziness was the most prevalent in MoA, MA, and CM (30%, 36.7%, and 43.4%, respectively; $p < 0.001$. The prevalence of vestibular symptoms was greater in MA and CM ($\chi^2 = 66.61$; $p < 0.001$). On the other hand, in MoA, the state of having no vestibular symptoms was more prevalent than any symptom (at least 10% higher; $p < 0.001$) (Table 1). Although the type of dizziness was different, its presence was not related to headache intensity ($p = 0.223$).

Analysis of the DHI scores showed that the degree of impact of dizziness did not differ among the groups ($p = 0.253$) (Table 1).

The presence of phonophobia and photophobia during the presence of vestibular symptoms had a greater prevalence in MA, followed by CM ($\chi^2 = 60.63$ and $\chi^2 = 61.87$; $p < 0.001$) (Table 2). The frequency of these migraine symptoms during dizziness followed the same order: higher in MA, followed by CM and MoA (Kruskal-Wallis' test: significance level = 0.000). The same pattern emerged when a headache occurred during the presence of vestibular symptoms (Table 1).

Concerning the ENG tests, calibration was regular in all evaluations; there were no differences in the presence of spontaneous (eyes open $p = 0.795$; eyes closed $p = 0.478$) and gaze nystagmus ($p = 0.516$). Although the patients were all young, only the smooth pursuit tests type I were considered normal, whereas types II and III were considered abnormal: MoA and MA presented more abnormal tests (53% and 70%, respectively) than CG (33%) and CM (47%;

TABLE 2. *Migraine symptoms associated with vestibular symptoms*

	CG (n = 30)	MoA (n = 30)	MA (n = 30)	CM (n = 30)	*p*
Phonophobia					
Never	0	5 (25%)	3 (10.7%)	5 (19.2%)	0.000*
Occasionally	0	7 (35%)	11 (39.3%)	12 (46.1%)	
Always	0	8 (40%)	14 (50%)	9 (34.6%)	
Photophobia					
Never	0	1 (5%)	2 (7.1%)	4 (15.4%)	0.000*
Occasionally	0	12 (60%)	9 (32.1%)	9 (34.6%)	
Always	0	7 (35%)	17 (60.7%)	13 (50%)	
Headache					
Never	3 (100%)	1 (5%)	0	0	0.000*
Occasionally	0	9 (45%)	15 (53.6%)	15 (57.7%)	
Always	0	10 (50%)	13 (46.4%)	11 (42.3%)	

Table entries are mean ± standard deviation or total (percentage).
*Significant *p* value.
CG indicates control group; CM, chronic migraine; MA, migraine with aura; MoA, migraine without aura.

TABLE 3. *Electronystagmography test results in CG, MoA, MA, and CM*

	CG (n = 30)	MoA (n = 30)	MA (n = 30)	CM (n = 30)	*p*
Calibration test					
Normal	30 (100%)	30 (100%)	30 (100%)	30 (100%)	
Abnormal	0	0	0	0	
Spontaneus nystagmus with eyes open					
Presence	1 (3.33%)	1 (3.33%)	0	1 (3.33%)	0.79504
Absence	29 (86.67%)	29 (86.67%)	30 (100%)	29 (86.67%)	
Spontaneus nystagmus with eyes closed					
Presence	4 (13.33%)	4 (13.33%)	8 (26.67%)	5 (16.67%)	0.47855
Absence	26 (86.67%)	26 (86.67%)	22 (73.33%)	25 (83.33%)	
Gaze nystagmus					
Presence	1 (3.33%)	4 (13.33%)	2 (6.67%)	2 (6.67%)	0.51592
Absence	29 (86.67%)	26 (86.67%)	28 (93.33%)	28 (93.33%)	
Smooth pursuit					
Normal (type I)	20 (73.33%)	14 (46.67%)	9 (30%)	16 (53.33%)	0.0390*
Abnormal (type II or III)	10 (33.33%)	16 (53.33%)	21 (70%)	14 (46.67%)	
Optokinetic test					
Symmetrical gain	22 (73.33%)	24 (80%)	23 (76.67%)	27 (90%)	0.4047
Asymmetrical gain	8 (26.67%)	6 (20%)	7 (23.33%)	3 (10%)	
Rotatory chair test					
Symmetrical response	27 (90%)	30 (100%)	29 (86.67%)	28 (93.33%)	0.3196
Asymmetrical response	3 (10%)	0	1 (3.33%)	2 (6.67%)	
Caloric test					
Normal function	28 (93.33%)	25 (83.33%)	28 (93.33%)	24 (80%)	0.2741
Vestibular weakness	2 (6.67%)	5 (16.67%)	2 (6.67%)	6 (20%)	
Electronystagmography results					
Normal	25 (83.33%)	12 (40%)	3 (10%)	8 (26.67%)	0.0000*
Peripheral etiology	1 (3.33%)	9 (30%)	7 (23.33%)	8 (26.67%)	
Central etiology	2 (6.67%)	2 (6.67%)	5 (16.67%)	3 (10%)	
Mixed etiology	2 (6.67%)	7 (23.33%)	15 (50%)	11 (36.67%)	

Table entries are total (percentage). Asterisk denotes a significant *p* value.
CG indicates control group; CM, chronic migraine; MA, migraine with aura; MoA, migraine without aura.

$p = 0.039$). At first sight, the optokinetic, rotatory chair, and caloric tests showed no differences among the groups (Table 3).

After the ENG tests were classified, CG and MoA had greater prevalence of normal tests ($\chi^2 = 40.96$; $p < 0.001$). MA and CM had higher prevalence of abnormal tests, with a predominance of mixed etiology (central and peripheral) ($\chi^2 = 40.96$; $p < 0.001$). When only abnormal tests in MoA were considered, alterations with peripheral etiology had a greater prevalence than alterations with central or mixed etiology ($\chi^2 = 40.96$; $p < 0.001$) (Table 3).

After a close look at each test performed during electronystagmography, we tried to identify whether any of the detected abnormalities modified the presence of vestibular symptoms. Considering only optokinetic tests with asymmetric gain, MoA, MA, and CM presented more tests with reduced gain (unilateral for MoA and MA and bilateral for CM) and higher incidence of vestibular symptoms; CG did not report dizziness ($p < 0.05$) (Table 4).

Besides, there were no differences in the number of asymmetric tests in rotatory chair tests among the groups, but there were differences in the variability of responses: MC (median = 13.5) and MA (median = 8.5) had higher measures than CG (median = 6.5) and MoA (median = 7; $p = 0.019$) (Fig. 1). There were no differences in the rotatory chair test responses with eyes open and closed ($p = 0.669$).

As for CP, there were no differences in caloric responses. In CG, MoA, MA, and CM, 36.7%, 30%, 30%, and 33.3% had asymmetric tests, respectively ($p = 0.936$). DP did not show differences among the groups, either: in CG, MoA, MA, and CM, 36.7%, 56.7%, 56.7% and 53.3% had abnormal results, respectively ($p = 0.347$).

DISCUSSION

Our results showed that MoA and MA presented more vestibular symptoms, mainly dizziness. Regarding migraine symptoms during vestibular symptoms, the presence of aura increased the co-occurrence of photophobia or phonophobia and vestibular attacks. MoA and MA presented more abnormal smooth pursuit tests (intensified in the presence of aura). The groups differed in terms of the distribution of symmetric measures at the rotatory chair test, with higher values in CM,

TABLE 4. *Presence of vestibular symptoms in asymmetric optokinetics for CG, MoA, MA, and CM among groups*

Optokinetic with Asymmetric Gain	CG (n = 8)	MoA (n = 6)	MA (n = 7)	CM (n = 3)	*p*
Vestibular symptoms					
Absence of vestibular symptoms	8 (100%)	2 (33.3%)	1 (14.3%)	0	0.017*
Presence of vestibular symptoms	0	4 (66.7%)	6 (85.7%)	3 (100%)	

Table entries are total (percentage).
CG indicates control group; CM, chronic migraine; MA, migraine with aura; MoA, migraine without aura.

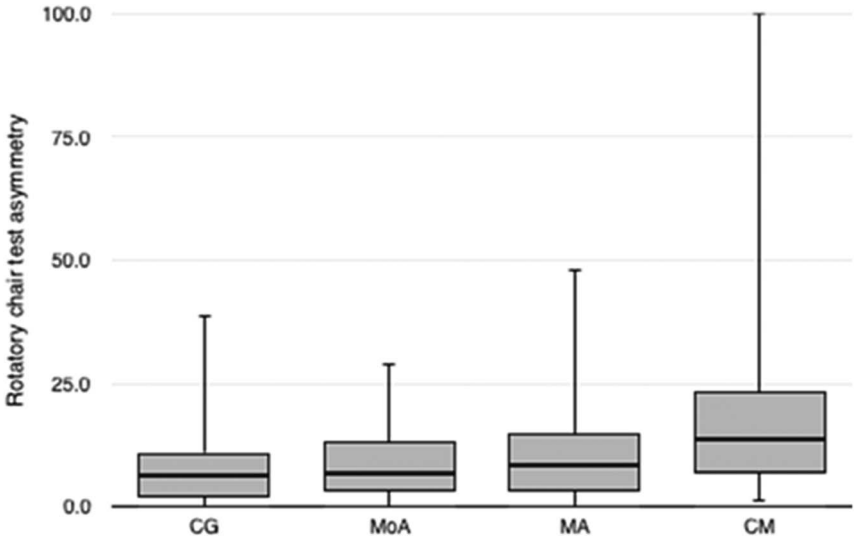

FIG. 1. Distribution of symmetry measures in rotatory chair test.

but not in terms of the caloric test responses. When optokinetic tests are asymmetric, there seems to be a tendency for developing vestibular symptoms. The tests showed that the vestibular function had more abnormalities in MA and CM, and these changes presented mixed etiology, peripheral and central.

Our findings support the hypothesis that migraine influences vestibular symptoms, which had a greater prevalence in MA and CM. Some studies have found a prevalence of vestibular symptoms ranging between 12% and 52%, with higher rates in the presence of aura (28,29). We found a greater prevalence of vestibular symptoms in migraine patients (66.6–93.3%) and a higher prevalence of spontaneous dizziness compared to spontaneous vertigo or triggered symptoms.

Previous studies have reported migraine symptoms associated with vestibular symptoms (15,30), but this relationship was demonstrated in patients who fulfilled vestibular migraine criteria (18). Other studies have suggested that migraine patients are more sensitive to light and sound even when they are headache-free (30). We assessed the presence of photophobia and phonophobia during vestibular symptoms in MoA, MA, and CM. Our results highlighted the close relationship between migraine and vestibular symptoms, with a greater prevalence of migraine and such symptoms happening together. The presence of headache during vestibular symptoms confirms this relationship and corroborates the hypothesis that inner ear structures are involved in the pathophysiological mechanisms of migraine, by facilitating central sensitization (31).

Experts recognize that the vestibular migraine (MV) classification leaves out many subtypes of the pathology, which avoids duplicate diagnoses (32). However, as already demonstrated, there is an overlap of several migraine and MV symptoms, not to mention that migraineurs can present different audiovestibular symptoms, making diagnosis difficult (17,32). In addition, patients with vertigo commonly have difficulty expressing themselves accurately, often not knowing how to differentiate between dizziness or imbalance and vertigo, which increases the likelihood of overlap or underdiagnosis (10).

To the best of our knowledge, this is the first study that has evaluated the vestibular function and symptoms in migraine subtypes and compared results between migraine groups and a control group. The electronystagmography findings in migraine patients are an exciting aspect of our study. Our data revealed that changes in the vestibular function were more prevalent in migraine patients (60–90%) and even more remarkable in MA and CM. In addition, vestibular function evaluation showed that MoA had higher prevalence of peripheral etiology changes, whereas MA and CM had higher prevalence of mixed changes (central and peripheral).

These changes in the ENG tests represented the sum of changes found in each test. In the optokinetic test, we found higher prevalence of vestibular symptoms in asymmetric tests, an evidence of this relationship's central component. The rotatory chair test and smooth pursuit results underscored this mixed etiology, mainly in MA, which had higher prevalence of pursuit types II or III and more variable asymmetry at the rotatory chair test. In each assessment, we found changes in tests that indicated central etiology and changes in tests that indicated peripheral etiology. For example, the oculomotor assessment was critical for indicating changes of central origin, along with changes of semicircular canal origin (peripheral) in the rotatory chair test and caloric tests. This confluence of changes within the same ENG test, indicating mixed changes, showed how migraine influences the vestibular system.

MoA, MA, CM, and CG did not differ in terms of caloric test: there were no statistical differences concerning vestibular weakness, CP, or DP. These data agreed with data reported in previous studies (33,34). Here, we assessed migraine subtypes. Compared to other studies, our results suggested no exclusive peripheral vestibular disorder.

The etiology of vestibular symptoms in migraine patients has been demonstrated in patients that fulfill the vestibular migraine criteria, indicating mixed or peripheral etiology (35,36). On the other hand, studies comparing migraineurs with or without vertigo have found abnormalities in the ENG

tests and vestibular-ocular reflex in both groups (37,38), but they have not shown differences among groups for the performed tests. The changes detected in these studies suggested that migraine acts on the vestibular system through different mechanisms and can trigger symptoms (38,39). Although our study stratified the groups in different ways (dividing the migraine subtypes to avoid overlapping diagnoses), our findings suggested the same: our ENG test results showed that migraine affects the vestibular pathways through different mechanisms, leading to tests with mixed etiological findings. These findings added to the variations we found in vestibular symptoms in the migraine subtypes and showed that the presence of aura and migraine chronicity intensified the influence of this pathology on the vestibular pathways.

There have been study findings supporting that migraine patients are more likely to present vestibular symptoms caused by stimuli than nonmigraineurs (40). These changes with mixed etiology highlight that migraine affects the vestibular system through different pathways, and that the presence of aura or chronic migraine intensifies these effects. Also, the present study supports the recommendation for finding a better instrument to measure vestibular symptoms in migraine patients given that DHI fails to quantify the impact of these symptoms (32). This implies the importance of clinically evaluating migraine patients: physicians should ask these patients not only regarding pain complaints but also regarding associated symptoms, such as dizziness, because migraine symptoms in patients with dizziness must be investigated.

With respect to the limitations of this study, we performed the ENG tests to evaluate the vestibular function, so evaluation involved oculomotor and lateral semicircular canal function assessment. More studies are needed to evaluate the vestibular function through other tests, in a less selective population. Another limitation regarding the ENG tests is that the caloric test stimulates the vestibular-ocular reflex in a lower frequency than the physiological frequency. The ENG test with caloric stimulation is widely used in clinical practice and the literature. Despite its limitations, it provides essential information for academic purposes and is highly applicable. A further limitation is that, because of our case-control design with no interventions, we can make no statement regarding causality. Nevertheless, the present study contributes to better understanding of the relationship between migraine and vestibular symptoms and of how it affects the balance system or postural control.

CONCLUSION

Our findings suggested that vertigo and dizziness progression with migraine chronicity and presence of aura is possible. Analysis of the optokinetic and rotational tests showed mixed vestibular impairment (central and peripheral). Although the response to the caloric test did not show any differences among the groups, its abnormalities overlapped with other ENG tests, highlighting that migraine can affect the vestibular system through different pathways.

The mechanisms behind functional vestibular changes in migraine remain unclear. However, mixed etiology in the ENG tests strengthens the influence of migraine on the vestibular system and the need for an early and multidisciplinary clinical approach for migraine patients.

REFERENCES

1. Stovner LJ, Hoff JM, Svalheim S, Gilhus NE. Neurological disorders in the global burden of disease 2010 study. *Acta Neurol Scand Suppl* 2014;198:1–6.
2. Woodhouse A, Drummond PD. Mechanisms of increased sensitivity to noise and light in migraine headache. *Cephalalgia* 1993;13:417–21.
3. Neuhauser HK, Radtke A, von Brevern M, et al. Migrainous vertigo: prevalence and impact on quality of life. *Neurology* 2006;67:1028–33.
4. So CW, Bent LR. Increased vestibular contribution to posture control in individuals with chronic headache. *J Vestib Res* 2009;19(1–2):49–58.
5. Bevilaqua Grossi D, Lipton RB, Bigal ME. Temporomandibular disorders and migraine chronification. *Curr Pain Headache Rep* 2009;13:314–8.
6. Florencio LL, Chaves TC, Carvalho GF, et al. Neck pain disability is related to the frequency of migraine attacks: a cross-sectional study. *Headache* 2014;54:1203–10.
7. Grossi DB, Chaves TC, Gonçalves MC, et al. Pressure pain threshold in the craniocervical muscles of women with episodic and chronic migraine: a controlled study. *Arq Neuropsiquiatr* 2011;69:607–12.
8. Bevilaqua-Grossi D, Pegoretti KS, Goncalves MC, et al. Cervical mobility in women with migraine. *Headache* 2009;49:726–31.
9. Ferreira MC, Bevilaqua-Grossi D, Dach FE, et al. Body posture changes in women with migraine with or without temporomandibular disorders. *Braz J Phys Ther* 2014;18:19–29.
10. Abouzari M, Goshtasbi K, Moshtaghi O, et al. Association between vestibular migraine and migraine headache: yet to explore. *Otol Neurotol* 2020;41:392–6.
11. Versino M, Mandalà M, Colnaghi S, et al. The integration of multisensory motion stimuli is impaired in vestibular migraine patients. *J Neurol* 2020; 267:2842–50.
12. Obermann M, Wurthmann S, Steinberg BS, et al. Central vestibular system modulation in vestibular migraine. *Cephalalgia* 2014;34:1053–61.
13. Ma Y, Zhang D, Wang W, Zhang Z, Zhang H. The comorbid mechanism of vestibular migraine and persistent postural-perceptual dizziness. *Lin Chung Er Bi Yan Hou Tou Jing Wai Ke Za Zhi* 2022;36:321–4.
14. Pereira CB, Nader SN, Kanashiro AK, de Carvalho WL. Vestibular migraine: vestibular symptom may identify different subgroups. *Otol Neurotol* 2016;37:281–3.
15. Beh SC, Masrour S, Smith SV, Friedman DI. The Spectrum of vestibular migraine: clinical features, triggers, and examination findings. Headache: the journal of head and face. *Pain* 2019;59:727–40.
16. Cutrer FM, Baloh RW. Migraine-associated dizziness. *Headache* 1992;32:300–4.
17. Benjamin T, Gillard D, Abouzari M, Djalilian HR, Sharon JD. Vestibular and auditory manifestations of migraine. *Curr Opin Neurol* 2022;35: 84–9.
18. Lempert T, Olesen J, Furman J, et al. Vestibular migraine: diagnostic criteria. *J Vestib Res* 2012;22:167–72.
19. O'Connell Ferster AP, Priesol AJ, Isildak H. The clinical manifestations of vestibular migraine: a review. *Auris Nasus Larynx* 2017;44:249–52.
20. Carvalho GF, Chaves TC, Dach F, et al. Influence of migraine and of migraine aura on balance and mobility—a controlled study. *Headache* 2013;53:1116–22.
21. Carvalho GF, Chaves TC, Gonçalves MC, et al. Comparison between neck pain disability and cervical range of motion in patients with episodic and chronic migraine: a cross-sectional study. *J Manip Physiol Ther* 2014; 37:641–6.
22. Carvalho GF, Vianna-Bell FH, Florencio LL, et al. Presence of vestibular symptoms and related disability in migraine with and without aura and chronic migraine. *Cephalalgia* 2019;39:29–37.
23. Harno H, Hirvonen T, Kaunisto MA, et al. Subclinical vestibulocerebellar dysfunction in migraine with and without aura. *Neurology* 2003;61: 1748–52.
24. Headache classification Committee of the International Headache Society. The international classification of headache disorders, 3rd edition. *Cephalalgia* 2018;38:1–211.
25. Jacobson GP, Newman CW. The development of the dizziness handicap inventory. *Arch Otolaryngol Head Neck Surg* 1990;116:424–7.

26. Ahmed MF. Standardization of rotatory chair velocity step and sinusoidal harmonic acceleration tests in adult population. *Med J Cairo Univ* 2014; 82:207–13.
27. Hallpike CS. Directional preponderance, 1942–1974. A review. *Acta Otolaryngol* 1975;79:409–18.
28. Calhoun AH, Ford S, Pruitt AP, et al. The point prevalence of dizziness or vertigo in migraine and factors that influence presentation. *Headache* 2011;51:1388–92.
29. Vukovic V, Plavec D, Galinovic I, et al. Prevalence of vertigo, dizziness, and migrainous vertigo in patients with migraine. *Headache* 2007;47: 1427–35.
30. Teggi R, Colombo B, Albera R, et al. Clinical features, familial history, and migraine precursors in patients with definite vestibular migraine: the VM-phenotypes projects. *Headache* 2018;58:534–44.
31. Lampl C, Rapoport A, Levin M, et al. Migraine and episodic Vertigo: a cohort survey study of their relationship. *J Headache Pain* 2019; 20:33.
32. Mallampalli MP, Rizk HG, Kheradmand A, et al. Care gaps and recommendations in vestibular migraine: an expert panel summit. *Front Neurol* 2022;12:812678.
33. von Brevern M, Zeise D, Neuhauser H, Clarke AH, Lempert T. Acute migrainous vertigo: clinical and oculographic findings. *Brain* 2005; 128:365–74.
34. Thompson-Harvey A, Mikulec AA. Total caloric eye speed in patients with vestibular migraine. *Am J Otolaryngol* 2018;39:180–3.
35. Yollu U, Uluduz DU, Yilmaz M, et al. Vestibular migraine screening in a migraine-diagnosed patient population, and assessment of vestibulocochlear function. *Clin Otolaryngol* 2017;42:225–33.
36. Celebisoy N, Gokcay F, Sirin H, et al. Migrainous vertigo: clinical, oculographic and posturographic findings. *Cephalalgia* 2008;28:72–7.
37. Baker B, Curtis A, Trueblood P, Vangsnes E. Vestibular functioning and migraine: comparing those with and without vertigo to a normal population. *J Laryngol Otol* 2013;127:1169–76.
38. Bir LS, Ardic FN, Kara CO, et al. Migraine patients with or without vertigo: comparison of clinical and electronystagmographic findings. *J Otolaryngol* 2003;32:234–8.
39. Altan Kayan J. Derrick Hood, neuro-otological manifestations of migraine. *Brain* 1984;107:1123–42.
40. Foster C, Pollard C. Comparison of caloric reactivity between migraineurs and non-migraineurs. *J Laryngol Otol* 2015;129:960–3.